tised. In advanced arterio-sclerosis every increase of the blood pressure which is the result of this treatment might lead to fatal consequences (embolism, apoplexy, rupture of aneurism of heart, or aorta). "Advanced sclerosis is, therefore, a contraindication for this treatment" (Schott). The value of many medicaments in angina comes from producing low blood pressure. The balneological and gymnastic treatment exercises a tonic influence, and "by strengthening heart muscle, as well as by acting on cardiac nerves, distressing symptoms of angina are either removed or reduced" (Schott).

Guidance should be had in regard to the bad effects of winds, great heat or cold, or rapid changes. Also, an atmosphere heavily laden with moisture is injurious. Internal conditions of dyspepsia and constipation must be warded against. A great deal of tact and good judgment are required, and the patient's disposition should be thoroughly known. Rest, particularly after meals, should be insisted upon, as patients are particularly liable to attacks at these times.

In general, supervision and counsel must be employed about exercise. Where an attack has lately occurred, it is wisdom to refrain from exertion for a while, especially if the heart is weak and fluttering, and afford it time to re-establish itself.

THE PATHOLOGY OF THE HEALED FIBROUS ADHESIONS OF THE PERICARDIUM.

By H. Gideon Wells, M.D.,
ASSOCIATE IN PATHOLOGY, UNIVERSITY OF CHICAGO.

In a total of 1048 autopsies upon the bodies of adults, the records of which are among those of the Pathological Laboratory of Rush Medical College (in affiliation with the University of Chicago) and of the Cook County Hospital, some inflammatory change was found in the pericardium in 128. These changes included everything in the nature of a pericarditis, from the frank serofibrinous and tuberculous processes to those with but a thin film of fibrin over a small area, or a fibrous band uniting the parietal and visceral layers at some point. Of these 128 cases 57 showed changes that were essentially chronic, the lesion consisting of fibrous tissue uniting the opposed surfaces for a greater or less extent. These figures agree closely with those of Leudet,[1] who found in 1003 autopsies adherent fibrous pericarditis 61 times. The remaining 71 were acute, and represented all the conditions usually seen in acute pericarditis.

Of the 57 cases with fibrous adhesions the cause could be attributed to rheumatism in 8; in 6 the lesion was frankly tuberculous, as shown by the presence of caseous masses or miliary tubercles in the adherent pericardium. There remain 43 cases of complete or partial synechia in which the cause cannot be thus determined from the anatomical findings or the history—certainly an impressively large proportion; 24 of these 43 were cases of total obliteration; in 14 the adhesions covered but part of the heart, and in 5 more a single firm band united the apex of the heart to the opposite pericardium. The large number of such cases of dry, fibrous synechia of the pericardium, for the most part lying latent during life and found only at autopsy, directly calls attention to the exactly similar condition that is still oftener found in the adjacent serous cavities, the pleural. Synechia of the pleural sacs, either total or partial, is an extremely common occurrence, and was recorded in 63 per cent. of all the 1048 autopsies from which these cases were gleaned, which figure is undoubtedly too low. In all but 3 of the 43 cases of "idiopathic" pericardial synechia there were fibrous pleural adhesions also present—that is, in 93 per cent. Furthermore, in a considerable number of cases in which the pleural adhesions were limited it was expressly noted that the pleura was adherent over the external surface of the pericardium. In several cases the pleural adhesions, where abundant, were expressly stated by the examiner to be especially firm over the external surface of the pericardium.

From these observations it would seem probable that the cause of adhesive fibrous pericarditis is in many cases the same as that of adhesive fibrous pleuritis. In addition, it is quite probable that the pericardial changes are secondary to those of the pleura much oftener than the reverse is true, for being a closed sac the pericardium has but limited chances of infection. The possible sources of infection of the pericardium are: 1. Through the blood stream. 2. Through the lymph vessels. 3. Direct infection by trauma. 4. By extension, which must be from (*a*) heart or vessels within the pericardium; (*b*) mediastinum; (*c*) pleura; (*d*) peritoneum.

Infection from the bloodvessels means a bacteræmia, which is bound most generally to produce an acute process in the pericardium, which is usually fatal and does not often terminate in the formation of fibrous adhesions unless the infecting agent be rheumatism, tuberculosis, or syphilis. Rheumatism affecting the pericardium usually affects the endocardium also, and these cases and other cases with a history plainly indicating rheumatic origin for the trouble have been excluded in this series. Of our 43 "idiopathic" cases, but two, so far as the history sheets tell us, had ever suffered from rheumatism. Syphilitic infection of the pericardium is one of the rarest of its manifestations, and when present is usually secondary to a gumma of the myocardium, which leaves sufficient traces to explain the source of any adhesions that might result. Mracek,[1] who has investigated the literature on this subject, says: "Syphilis has hardly ever caused an exudative serofibrinous pericarditis." A. Genersich[1] has recently reported such a case. Nor does syphilis often affect the pleura. Among the autopsies studied there were 35 cases of marked syphilis, but in none were the pleura or pericardium evidently affected by it. Tubercle bacilli reaching the pericardium, or any other place, by the bloodvessels produce almost necessarily distinct tubercles, so that should the resulting pericarditis heal—which cannot be common in a tuberculosis of vascular origin—the characteristic fibroid, caseous or calcareous nodules would usually give evidence of the source of the adhesions.

From the lymph vessels the most common infection would be tuberculous. This is favored by the fact that the lymphatics of the pericardium empty, in common with those of the pleura, either directly or *via* the glands at the base of the pericardium, into the glands about the large bronchi and the root of the aorta. The frequency with which tuberculous lesions affect these glands makes them the possible source of pericardial infection. To be sure, in thus explaining infection of the pericardium we must invoke a reversal of the normal current in the lymph vessels; but this phenomenon has been shown to occur by v. Recklinghausen,[2] and its results have been observed especially in the transmission of malignant neoplasms (*vide* article by Witte[3] on "Retrogressive Lymphatic Transportation of Carcinoma"). The choked condition of the lymph spaces in the tuberculous peribronchial glands would favor such a reversal of the direction of the lymph flow. Here, again, we would expect to find, at least frequently, miliary tubercles or their remains, as in vascular tuberculosis; but a lymphatic tuberculosis is likely to be a much more trivial affair, recoverable, and leading often to a simple sclerosis of the perilymphatic tissue. Hence it is possible that an adhesive pericarditis might result in this way, extensive or limited, and a few years after recovery no morphological evidences of its tuberculous nature remain. The connection of such a pericarditis with fibrous pleuritis is plain.

Direct infection of the pericardium by trauma can scarcely come under consideration in this matter of simple fibrous pericarditis. The occurrence of a fibrous mediastino-pericarditis due to repeated pressure on the sternum in certain trades, while affirmed by a few, is not generally credited. Roberts[3] states that he has met with no such cases. M. Labbé[4] reports a case of fatal cardiac incompetence following a pericarditis which he considered due to a blow on the chest. Such instances are extremely rare.

Of sources of direct extension the heart and the parts of the large vessels within the pericardium must be least common, since they are so rarely the seat of any extensive inflammatory processes. No relation between the simple diffuse chronic myocarditis and pericarditis could be found in the cases studied, except in those cases in which the pericarditis, being primary, had affected the underlying myocardium.' Localized myocarditis, acute or tuberculous, would leave sufficient evidences to determine the source of the secondary pericarditis. From the mediastinum infection might travel readily enough to and into the pericardium. Except in the lymph glands, infections in this space are rare, and a resulting process in the pericardium would have its course plainly indicated. But in the mediastinal lymph glands we have probably the commonest site of quiescent tuberculosis. These glands are in the closest relation to the pericardium possible, especially over its base. Another place of close contact is where the pericardium is carried along the left branch of the pulmonary artery, passing between the artery and the left bronchus. A gland seems to lie between this extension of the pericardium and the bronchus quite constantly. The small glands of the anterior mediastinum are also to be remembered, as they are often tuberculous. There can be no question of the relationship of these glands to tuberculosis of the pericardium. This relation has been consistently mentioned by every writer on tuberculous pericarditis, but the manner of infection has not been so clearly indicated. The possibility of dissemination of the bacilli by way of the lymphatics has already been considered. Occasionally a caseating gland breaks into the sac and erupts into this space, resulting in a rapid, caseating, or purulent inflammation of the entire surface. Such cases have been reported by Kast[1] and Mickle.[4] But generally the mechanism of the infection is not so evident. Usually it is simply found that a number of caseous glands lie upon the external surface of the pericardium, united to it by adhesions which sometimes are dense, but often only slight, and there is no continuity of the tuberculous lesions of the gland and those of the sac. In some cases the inflammation between the membranes is typically tuberculous, but in many it presents simply a mass of fibrous tissue of varying thickness, and neither macroscopically nor microscopically can a single feature warranting a diagnosis of tuberculosis be found. Such a case is the following;

CASE I.—J. M. G., colored, male, aged thirty-two years. Died with a general anasarca and marked albuminuria, giving rise to the diagnosis of chronic parenchymatous nephritis. No history of rheumatism or other acute inflammatory process was obtained. At autopsy the diagnosis was sustained; but, in addition, an extensive tuberculosis was found, with caseous and ulcerative lesions in the lungs, intestinal ulcers, and caseous and miliary tubercles in the abdominal and mediastinal lymph glands. There was also found an extensive bilateral fibrous pleuritis and a gray thrombus in the right internal jugular vein. In the right auricular appendix was a small mixed thrombus infiltrated with leucocytes. Both layers of the pericardium were thickened about equally, each being perhaps 2 mm. thick, and uniting them throughout was a layer of soft, bluish-gray connective tissue, which was easily torn, though firmer at the base than lower down. Nowhere could anything resembling tuberculous lesions be found in the pericardium or in the exudate. The glands at the base of the pericardium, particularly over the right auricle, were closely adherent to the external surface of the membrane; but a careful dissection of these glands, supplemented by microscopical examination of the intervening tissues between these glands and the pericardium failed to show any extension of the tuberculous lesions outside of the gland capsule. A piece of the pericardium, with the soft exudate, was placed in the abdominal cavity of a guinea-pig; the animal remained in good health, and, when killed, eighty days later, showed no tuberculous lesions. Sections were made through many parts of the pericardium and examined microscopically, always negatively as to any lesions resembling those of tuberculosis. Everywhere the structure was that of a poorly vascularized, new-formed connective tissue, with few cells, which nowhere showed any tendency toward grouping. A few sections were stained for tubercle bacilli, with negative results. On the contrary, the lymph glands immediately adjacent to the pericardium showed all the characteristics of tuberculosis.

This illustrates a case which is almost certainly tuberculous, the origin probably from the glands in contact with the pericardium, but in which the manner of extension to the inner surface of the sac cannot be found, and in which the resulting lesion bears none of the characteristic anatomical evidences of its tuberculous origin. This case is but one of a number. Of the twenty-four cases of total obliteration of unexplained etiology, tuberculous lesions of the glands immediately adjacent to the pericardium were noted in ten. Inability to locate any direct extension from the tuberculous glands to the pericardium was likewise the result in two cases of tuberculous pericarditis, with abundant characteristic nodular lesions, that I have examined.

From this it is evident that a condition of total or partial synechia of the pericardium may and often does come from tuberculous mediastinal glands, without the healed lesions of the pericardium bearing any traces of their tuberculous origin. Two possible explanations suggest themselves. One is that the changes in the pericardium, although due to infection by the specific organism, at no time presented the anatomical characteristics of such an infection. It is by no means rare for tuberculosis in other localities to fail to produce the usual structural changes, and this is particularly true of serous surfaces. A. N. Perón[1] has shown that in tuberculous pleurisy the lesions in the early stages are not different from those of a simple inflammation except for the presence of the bacillus, and even later distinctive tubercles often do not form, so that in the healed condition there is no evidence of tuberculosis. The second explanation is derived from a comparison with the conditions about tuberculous glands other than those of the mediastinum. It is well known that the diseased glands of a chain in the neck are frequently fused by dense masses of firm fibrous tissue. Microscopical examination of this shows it to consist simply of fibrous tissue, poor in cells, and nowhere presenting any of the evidences of tuberculosis. The capsule of the glands is found to be outside of all characteristically tuberculous cellular groupings. On the other hand, this process of sclerosis may extend for considerable distances from the nearest tuberculous glands. Since the sclerosis is seen outside of glands that are still increasing in size, it cannot be considered as the results of a healed tuberculosis. A distinct resemblance between this process and that seen in the pericardium is evident. The explanation that is most reasonable, and one quite generally accepted, is that the periglandular fibrous hyperplasia is due to the action of the products of the tubercle bacilli that have diffused outward into the tissues. The sclerogenic action of tubercle toxin is marked, especially when it is so diluted or so composed that the necrogenic action is excluded. J. Auclair[10] claims to have been able to obtain from cultures of tubercle bacilli two different substances, one soluble in ether, the other in chloroform, the first of which produces the necrogenic effects, the other only the sclerogenic. It would seem quite reasonable to assume that a similar diffusion of sclerogenic substances occurring from the peribronchial and other mediastinal glands would invade the pericardium or pleura and lead to a simple connective-tissue formation in these cavities. In favor of this view is the fact that the two sacs and the mediastinal tissues are often found fused together by a mass of firm fibrous tissue enclosing tuberculous glands; and, further, that in many of the specimens of fibroplastic pericarditis the lesions are definitely older over the base—*i. e.*, in close proximity to the glands, than elsewhere, as shown in Case I.

The pleura is probably the most frequent source of non-tuberculous pericarditis, except for the rheumatic form, if we consider the frequency of pericarditis in pneumonia, which caused twenty-nine of the seventy-one acute cases found at autopsy. It is not to be denied that rheumatism is a more common cause clinically, but the rheumatic cases do not often come to autopsy in the acute stage. Pneumococcus pericarditis, while perhaps sometimes hæmatogenic, is oftenest produced by direct extension from the pleura. It is not uncommon to see the inflammatory process just beginning over the inner surface of the pericardium in closest relation to the inflamed pleura. Such a process, healing, might leave pleural and pericardial adhesions; undoubtedly it sometimes does. But it is universally admitted that tuberculosis is the most frequent cause of pleural inflammation, and it seems proper to assume that many of the pericardial adhesions result from the extension of this process through the pleuropericardial wall. This extension may be by growth of the tubercles through the wall, or perhaps by diffusion of the sclerogenic products of the organism. A roundabout route may also be assumed, the mediastinal glands becoming infected from the pleura, and in turn infecting the pericardium in the ways before mentioned.

Occasionally cases of tuberculous peritonitis are accompanied by tuberculous pericarditis, which seems due to an extension through the lymph vessels of the diaphragm. More often this condition is accompanied by tuberculosis of the pleura, sometimes also of the meninges and joints, seeming to be a matter of a general tuberculous serositis. Rarely a simple peritonitis produces inflammation of the pericardium. None of the cases in this series, however, gave evidence of relation to the peritoneum.

By the above process of exclusion it has been attempted to show that *many cases of adherent pericardium are probably of tuberculous origin, although not showing any anatomical characteristics of tuberculosis.* However, it is to be understood that this is not the only cause. Many other affections of the pericardium may heal and leave adhesions, and this may occur as well in an individual with tuberculous peribronchial glands as in anyone else, so the existence of caseous or calcified glands is not conclusive evidence that adhesions found in the pericardial sac are due to tuberculosis. However, when the patient's history shows freedom from rheumatism, pneumonia, and other acute processes, and autopsy reveals a pericardial synechia accompanied by a similar condition in the pleural cavity, or healed or active tuberculosis in the mediastinal glands, or both, the assumption that tuberculosis has caused the pericardial adhesions cannot often be wrong.

Returning to the forty-three cases of fibrous pericarditis in which no etiology has been assigned, it is found that forty showed pleuritis, and in eighteen healed tuberculosis was demonstrated. Of these eighteen it seems proper to consider tuberculosis the most probable cause of the pericarditis in thirteen, for the aforementioned reason. The following cases are cited to illustrate these points:

CASE II.—A negress, aged thirty-five years, who died of uræmia, was found to have, in addition to an advanced chronic nephritis, a total obliteration of the pericardial cavity by firm fibrous adhesions. Both pleural cavities were similarly obliterated. In the lungs were a few small areas of caseating tuberculosis near the apices, and a few miliary nodules were present in the spleen. There was a general tuberculous lymphadenitis, affecting principally the mesenteric, retroperitoneal, anterior mediastinal, and peribronchial glands. The glands about the pericardium were intimately adherent to it. No caseation, miliary nodules, or other evidences of tuberculosis were seen in the layers of the adhesions. The heart weighed 440 grammes, the enlargement being in the left ventricle, and explained by the renal disorder.

CASE III.—A man, aged twenty-four years; death from phthisis and amyloidosis of the abdominal viscera. The pericardium was adherent over the entire surface of the heart, the adhesions showing no caseous or other distinctly tuberculous alterations. Both pleural cavities were likewise obliterated, and the lungs were firmly bound to the external surface of the pericardium. Advanced ulcerative and nodular tuberculosis affected both lungs extensively. The peribronchial lymph glands were much enlarged and contained tuberculous areas. It is recorded that the heart was "somewhat enlarged," but there are no details as to the changes.

CASE IV.—Man, aged sixty-six years; death from general tuberculosis of pulmonary origin. Both pleural cavities obliterated by fibrous adhesions, and the lungs thus bound to the pericardium. Uniting the heart to the pericardium beneath the adherent pleura is a band of fibrous tissue.

Of the twenty-two remaining cases of fibrous synechia in the series without assignable etiology there are fifteen accompanied by a fibrous pleuritis, without other anatomical changes being found to explain the pericardial adhesions. Of the other seven a ready explanation is found in four. One was due to extension of a sarcoma, two to extension of aortic aneurism, and one from a healed myocardial inflammation.

The cases accompanying fibrous pleuritis, and without indications of the cause of either the pleuritis or the pericarditis, are fourteen in number. Of these only the following are of special interest:

CASE V.—Man, aged thirty-eight years; died from asystole. The clinical diagnosis was: Chronic adhesive pericarditis; cardiac hypertrophy, with dilatation; pressure on recurrent laryngeal nerve, with resulting paralysis. At autopsy the obliteration of the pericardial cavity by adhesions was found to be complete, and externally it was firmly united to the pleura and to the diaphragm. The heart was hypertrophied, there was chronic mitral and tricuspid endocarditis, and both auricles and the right ventricle were dilated. General passive congestion, with anasarca, was present, and a renal infarct was found, the embolus arising in a thrombus in the left auricle. Both pleural cavities were obliterated by firm adhesions, and where the left recurrent laryngeal nerve passed beneath the aorta it was compressed for a distance of one-half an inch by dense fibrous tissue. No evidences of tuberculosis were found, and the history states that the patient had not had rheumatism or other acute infection. This case was reported by Dr. J. B. Herrick[11] before the Chicago Medical Society, February 9, 1898.

CASE VI.—Male, aged fifty-seven years; death from asystole, secondary to chronic interstitial nephritis, arterio-sclerosis, and myocarditis. A few thin, fibrous adhesions passed from the right ventricle to the pericardium. A few fibrous adhesions were present in the pleural cavities. No tuberculosis. In the left auricular appendix was a mixed thrombus.

CASE VII.—Man, aged sixty years; died from cardiac failure. The pericardial cavity was obliterated by firm, old adhesions. Adhesions were abundant and strong in the pleural cavities. No tuberculosis. Clinically, the case was diagnosed as chronic endocarditis, but at autopsy there were no changes in the valves, although the heart was hypertrophied and dilated.

CASE VIII.—Man, aged sixty-one years; death from an uncompensated aortic stenosis. Adhesions obliterated the pericardial cavity over the base of the heart. In the left auricle was a mixed thrombus. Both pleural cavities contained fibrous adhesions, and in the right apex were calcareous nodules.

There were four cases in which no explanation whatever of the finding was possible, there being no healed tuberculous or fibrous pleuritis, nor yet any history of any acute infectious process. There are, therefore, some eighteen cases of fibrous pericarditis in which it is impossible to find the cause, except that in all but four it was associated with more or less extensive similar changes in the pleural sacs. To attempt to state any one thing as the cause in any of these cases would be mere guesswork; but the frequency of the co-existence of pleuritis is, perhaps, of some significance. If we are to consider the cause of the two conditions as the same, the occurrence of fibrous pleuritis as the result of tuberculosis without recognizable lesions must be considered, and also the relation to previous pneumonia and simple acute pleuritis. Nephritis accompanied many of these cases, and its relation to fibrous pericarditis, although doubtful, is of interest. Serous inflammations in nephritis generally mark the termination of the chronic interstitial affection, and Bosc[12] seems to consider it as altogether a terminal occurrence. He states that it does not seem capable of regression, and is followed by death in some days, or, rather, it is present at the moment of death. On the other hand, W. Ewart[13] believes that effusion may occur transiently, without evidences of acute pericarditis, in Bright's disease, as well as in rheumatism and cardiac troubles; usually this is of mechanical origin, but, as in the case of analogous pleural effusions, they may be dependent upon a subacute inflammation. If Ewart's ideas are correct, then nephritic pericarditis may occasionally be recoverable, and adhesions may originate in this way. But allowing for uræmia, pneumonia, and all the evident sources of pericardial adhesion, there remain a few cases the origin of which can only be conjectured from the anatomical findings. In these cases the history is often of help, giving evidence of pneumonia, rheumatism, acute infections of other sorts, which offer suggestions, while not permitting a positive conclusion.

RHEUMATIC PERICARDITIS. Eight cases of fibrous synechia in this series were directly attributable to rheumatic pericarditis, the following being illustrative:

CASE IX.—L. G., aged fifteen years, female; St. Elizabeth's Hospital, service of Dr. Sanger Brown, autopsy by Dr. Le Count. This girl entered the hospital with severe chorea and acute articular rheumatism. Death resulted from asystole. At autopsy general œdema and passive congestion of cardiac incompetence were marked. The heart was greatly enlarged, weighing 840 grammes with the adherent pericardium, and the cavities were much dilated. The mitral valves were incompetent, and the aortic cusps were the seat of a recent verrucous endocarditis. The pleural cavities contained no adhesions, and no traces of tuberculosis could be found either in the lungs or in the lymph glands. Although the peribronchial glands were enlarged and soft, they contained nothing that could be interpreted as tubercles. The pericardial sac was completely obliterated by firm adhesions, which when torn left a reddish surface, sprinkled with small grayish and yellowish points, some of which protruded. This appearance was identical with that of an acute miliary tuberculosis. Microscopically, the "tubercles" were found to correspond to small foci of round cells embedded in the adhesions, which nowhere had the structure of tubercles, although many pieces from different parts of the heart were examined. A piece of this tissue placed in the abdominal cavity of a guinea-pig did not produce tuberculosis, and sections stained for tubercle bacilli did not reveal their presence. Sections of the lymph glands showed the enlargement to be due to a simple endothelial and connective-tissue hyperplasia, with no evidences of tuberculosis.

Case X.—Man, aged twenty-five years; died with asystole. Five months before death this patient had an attack of acute articular rheumatism, and one month later the cardiac symptoms became manifest. The clinical diagnosis was chronic endocarditis, cardiac hypertrophy, and acute dilatation. On opening the thoracic cavity the pericardium was found to present an unusually large surface, extending 10 cm. to the left and 6 cm. to the right of the median line. On the left side there were a few adhesions between the lungs and the pericardium, but *there were no adhesions to the chest wall.* On opening the pericardium it was found that although the external layer was not noticeably thickened it was united to the epicardium by firm fibrous adhesions, separated with difficulty. The heart was much enlarged, weighing 700 grammes; the ventricles were dilated, the right being 11 cm. in depth, the left 10½ cm. Both auriculo-ventricular orifices were much enlarged. The myocardium showed no macroscopical changes. All the valvular surfaces were normal. General anasarca and passive congestion of the lungs, liver, spleen, and kidneys gave evidence of the valvular incompetence. No evidences of tuberculosis could be found in the lungs, pleura, glands, or elsewhere. There were no pleural adhesions except those uniting the left lung to the pericardium, which presumably originated from the latter.

Case XI.—A woman, aged twenty-two years; death from asystole, with the following clinical diagnosis: Mitral regurgitation and stenosis, dilatation of the heart, double hydrothorax, hydroperitoneum, thrombosis of the left axillary vein. Her trouble all dated to an attack of rheumatism five weeks before death, with evidences of involvement of the heart, although no positive evidences of pericarditis were obtained while she was in the hospital. Three years previously she had had an attack of articular rheumatism, not accompanied or followed by cardiac manifestations. The anatomical diagnosis was as follows: Acute mitral and aortic endocarditis; mitral stenosis; hypertrophy and dilatation of the heart; thrombosis of the left auricle and of the left subclavian and axillary veins; general anasarca; healed splenic infarct; passive congestion of the liver and spleen; fibrous internal and external pericarditis. In this case the external surface of the pericardium was found adherent to both lungs where they were in contact with it, and over the apex to the chest wall. The cavity was completely obliterated by very firm fibrous adhesions, showing no nodules, caseous or fibrous. No adhesions were found in the pleural cavity except those about the pericardium, and the glands and lungs showed no evidences of tuberculosis.

Case XII.—Man, aged thirty-five years. Clinical diagnosis: Cerebral embolism, chronic degeneration of the kidneys, left heart hypertrophy, œdema of the lungs. Anatomical diagnosis: Chronic mitral, tricuspid, and aortic endocarditis; hypertrophy and dilatation of the heart; softening of the left basal ganglia and left cerebral cortex; renal infarct; general passive congestion; cirrhosis of the liver; chronic fibrous pericarditis; and localized fibrous pleuritis. On the outer surface the pericardium was attached to the left lung by one fibrous band; internally the cavity was totally obliterated by firm fibrous adhesions, simple in character. In this case cardiac symptoms were first observed during an attack of rheumatism four months before death, with dyspnœa, which had been marked up to the time of death, and by cerebral embolism.

CASE XIII.—Woman, aged forty-five years, dying with asystole. Anatomical diagnosis as follows: Acute and chronic mitral and aortic endocarditis; mitral stenosis; thrombus in right auricular appendix; infarcts in the lungs, with localized fibrinous pleuritis; general passive congestion; obliterative fibrous pericarditis. The pericardial sac was obliterated by simple fibrous adhesions, and externally adherent to the right lung. No tuberculosis. This patient had repeated attacks of rheumatism during the last twenty years of her life. First noticed cardiac symptoms twelve weeks before death, after exposure to cold, which exposure resulted in no other trouble besides that of the heart.

Comparing these cases with those of tuberculous origin, the following anatomical differences are seen: *In the rheumatic there is a total absence of tuberculous lesions in the mediastinal glands and lungs; the pleuritis existing is generally localized at the pleuropericardial surfaces, seeming to be secondary to the pericarditis; endocardial changes are usually present, in all but one of those recorded; death is usually due to cardiac failure in the rheumatic, while this is the exception in the healed tuberculous pericarditis; the age of the subjects is, on the whole, younger in the rheumatic.* So far as the changes in the pericardium are concerned, they differ not at all from those of the simple fibroplastic changes sometimes produced by tuberculosis, as described previously. On the other hand, in one case (Case IX.) the changes resembled macroscopically so closely those of a miliary tuberculosis of the pericardium, with sclerotic changes predominating, that it required considerable microscopical and experimental evidence to exclude tuberculosis. It is of interest that all but the first of these eight cases was classified as of probable rheumatic origin on the autopsy findings alone, and the verification of this diagnosis by the history when subsequently investigated showed that the differences above described are sufficiently characteristic to be of value. The relative effects of rheumatic and tuberculous pericarditis on the heart itself, which are of much clinical interest, will be discussed later.

CALCIFIC PERICARDITIS. Of this rather unusual form of pericardial lesion there are four examples found in this series.

CASE XIV.—Man, aged thirty-five years; death from chronic nephritis. The pericardial change evidently attracted the attention of the examiner, for the clinical diagnosis made before autopsy reads: "Chronic nephritis; pleurisy, with effusion; pericarditis or mediastinal tumor." The anatomical findings were as follows: General anasarca; fibrous pleuritis and peritonitis; fibrous pericarditis, with calcareous plates; serofibrinous pleuritis; dilatation and hypertrophy of the heart; interstitial pneumonia; cirrhosis of the liver, with congestion of the spleen and gastro-intestinal tract; chronic diffuse nephritis. Externally the pericardium was bound firmly to the diaphragm and lungs; internally it was found obliterated completely, and embedded in the adhesions were large, thick, calcareous plates. No tuberculosis found anywhere.

CASE XV.—A "middle-aged" woman, who died after an operation for empyema. Unfortunately the record of this case is incomplete, it having been done at a private autopsy outside the hospital, and it is impossible to improve the data. The anatomical diagnosis reads: Right pleuritis and thoracotomy wound; left fibrinous pleuritis; adhesive fibrous pericarditis, with calcareous plates; atrophic cirrhosis, with passive congestion of the spleen. It is stated that the pericardial layers were adherent, and in the adhesions were found irregular, calcareous masses; one, on the anterior surface, measured 3 cm. by 2 cm.; on the left border was one one-half the size of the fist; over the diaphragmatic surface was one 2 cm. in diameter. The heart was the seat of some hypertrophy and more marked dilatation, the left ventricle measuring 0.5 cm. in depth. No tuberculous changes were found in the lungs. Here the pericardial change seems definitely older than in the pleura, for the latter is described as fibrinous.

CASE XVI.—A man, aged thirty-five years, who died from asystole, with the following clinical diagnosis: General arterio-sclerosis; chronic endocarditis; insufficiency of the mitral valves; chronic parenchymatous nephritis. The anatomical diagnosis: Chronic adhesive pericarditis, with calcification; chronic mitral endocarditis, with stenosis; hypertrophy and dilatation of the heart and tricuspid incompetence; chronic adhesive pleuritis; general passive congestion, with slight anasarca; multilobular cirrhosis; diffuse nephritis. The pericardial sac was completely obliterated; there were calcareous masses in the right and upper border and a large collection of "stony masses" in the sac proper. Externally it was adherent to the left lung. No tuberculosis found in the body.

CASE XVII.—Man, aged fifty-five years, who died of acute nephritis. At autopsy were found: Chronic fibrous pleuritis; chronic fibrous pericarditis, with calcification; atheroma of the aorta; acute parenchymatous nephritis; cirrhosis of the liver, with jaundice; acute myocarditis, with myocardial segmentation. The pericardial cavity was found completely obliterated by friable fibrous adhesions, greatly thickened, and between the layers of the pericardium toward the base was a large calcareous plate. There were no changes in the lungs, and traces of tuberculosis were not found.

Calcification of pericardial exudate is rare enough to be of considerable interest, although the occurrence of four cases in such a relatively small number of autopsies leads to the belief that its rarity lies chiefly in the infrequency with which it has been reported. In 1899 Fritz Diemer[14] was able to collect but ten cases, beside two of his own, in which the calcification was extensive, more or less completely covering some part of the heart. Three of the four cases mentioned would come under this description. Perhaps the most remarkable instance of this condition was reported in this country in 1890 by Drummond.[15] In this case the greater part of the heart was surrounded by a stony covering; the right ventricle had to be opened with a saw. The calcification extended through the right auricle and through the entire thickness of the left ventricle. It varied from one-quarter to one inch in thickness, and seemed to involve the epicardium only, not the parietal layer, which could be stripped off.

The etiology of this condition is very obscure. Diemer states that in his twelve cases it was hinted at in six only. His series certainly throws no light on the subject, except in pointing out the fact that in only one was any tuberculous lesion found, and that not related to the pericardium. We know that calcification occurs only in dead or dying tissues, or inspissated exudates, which, from analogy, are probably purulent. Calcification of adhesions is seemingly the less common of the two. In all the four cases described in this series it seems most probable that these were instances of calcified inspissated exudates. What form of exudative pericarditis is most likely to heal and leave the proper conditions for calcification? Certainly not tuberculous, for a caseating tuberculosis of the pericardium would seldom last long enough for this, and if it did it would be accompanied by characteristic lesions locally or remotely; but in none of these four cases, and in only one of Diemer's twelve was tuberculosis found anywhere in the body. However, Püschmann[16] has reported a case of calcareous myocarditis of tuberculous origin. The purulent exudate which is least malignant and most prone to become sterile and inspissate is that arising in pneumococcus serositis, and, further, this is one of the most common causes of acute pericarditis. It also agrees with the conditions observed in most cases of calcareous pericardium, in usually leaving no traces of its origin in the lung except fibrous pleuritis. In the first case of this series the existence of a marked interstitial pneumonia is of significance, as also the pleural thickening in three of Diemer's cases. Case XV. gave a history of pneumonia eleven years before death, and also of attacks of rheumatism, the last two years before death. Unfortunately, in the other cases the history is incomplete. Ewart[17] has reported a case of extensive calcification of the pericardial adhesions resulting from rheumatism.

A most important as well as interesting observation is that *in each and every one of these four cases of calcareous pericarditis a marked cirrhosis of the liver was present.* On looking over Diemer's[11] cases it is found that in two of the twelve no reference as to the liver can be obtained. Of the remaining ten, in seven a distinct atrophic cirrhosis was observed; in one a fibrous perihepatitis, combined with advanced syphilitic cirrhosis, was found; in another a fibrous perihepatitis alone; in but one was there no fibrous change in the liver. This striking feature seems to have been overlooked by Diemer. In only one of these nine was any reference made to alcoholism, this individual being rated as a moderate drinker; in one case—that of Drummond[11]—the macroscopical appearance of the liver suggested tuberculous cirrhosis, but this was not confirmed microscopically. In the remaining cases there is nothing to explain this cirrhosis. Further reference will be made to this matter later in considering the results of pericardial synechia.

RESULTS OF PERICARDIAL ADHESIONS. For the most part, synechia of the pericardium seems to be altogether without effect on the patient, and it is not usually recognizable during life, even in the cases in which it does produce some effects that are readily referable to the heart or circulatory system. Like the fibrous pleuritis, it is essentially an autopsy finding. Of the fifty-seven cases of fibrous pericarditis, of whatever nature, in the entire series, in thirty-six of which the pericardial obliteration was total, the condition was recognized during life in but three. For the consideration of the effects of pericardial adhesions the cases may be conveniently divided into three classes:

1. Those with but one or a few bands of fibrous tissue connecting the parietal and visceral layers.
2. Complete or nearly complete obliteration of the pericardial sac, without adhesions between the external surface and the surrounding structures.
3. Pericardial sac completely obliterated and adherent externally to the adjacent structures—that is, combined internal and external pericarditis.

It is hardly probable that a few bands such as are often seen lying upon the auricles, or attaching the apex of the heart to the parietal pericardium, interfere in any appreciable degree with the action of the heart.

More extensive adhesion may, and in many cases most certainly does, prevent perfect cardiac function. It is quite generally recognized that in the third group of cases, in which the heart is bound to the pericardium, and this in turn to the chest wall, the lungs, the posterior mediastinum, and the diaphragm, that cardiac dilatation, hypertrophy, and ultimately incompetence often result. This process is seen particularly in rheumatic cases, and is one of the most rapidly fatal of the cardiac complications of rheumatism, if not the most so. From the nature of the changes present, an incompetence established in this way is absolutely unaffected by treatment, and the course is rapidly and progressively downward. Pericardial obliteration from causes other than rheumatism less often leads to cardiac failure. It will be recalled that in the cases of this series ascribed to tuberculosis there were no deaths at all from asystole, whereas in those of rheumatic origin the cause of death lay in the heart alone, either from the pericardial lesion or from valvular changes, or both, in all but one, which terminated prematurely from cerebral embolism (Case XII.). English writers have considered this matter of asystole of pericardial origin most extensively, and ascribed to it the proper importance. The reason that the rheumatic pericarditis is so much more prone to cause fatal incompetence of the heart is ascribed by them quite generally to changes occurring in the myocardium during the rheumatism; in other words, rheumatic pericarditis is generally a pancarditis affecting valves, muscles, and pericardium together. From this condition dilatation results, and Dickenson[18] argues that the chief effect of the pericardial adhesion is to prevent this dilatation from being overcome as the heart gains strength—*i. e.*, the dilatation is primary, and not due to the adhesions. Hypertrophy follows, but under disadvantages, and compensation is never attained. "Thus it is," he says, "that adhesions following upon non-rheumatic pericarditis, which even in children is attended by little enfeeblement of the heart, proves generally harmless."

While not questioning in any way the logic of Dickenson's reasoning, nor doubting the correctness of the inference, there is another factor that does not seem to have been brought forward. This is based upon the function of the pericardium as determined by H. L. Barnard.[19] Barnard has shown that its function is, beyond that of providing a smooth surface to permit motion, to limit dilatation of the heart and to relieve strain upon the muscle, especially upon the right ventricle during diastole. He compares its function in relation to the heart muscle to that which the fibrous adventitia of the arteries bears to the muscular coat. First, he found that normal pericardium is practically inextensible. Then he demonstrated that, while the heart of a dog within the pericardial sac ruptured under a minimum pressure of 1¼ to 1¾ atmospheres, when freed from the pericardium it ruptured from a pressure of ¾ to 1 atmosphere. Rupture within the pericardium is preceded by a rupture of that sac, which gives way at its attachment to the great vessels. (Barnard says that this part of the pericardium seems to be the only part that contains nerves, and suggests that this may bear some relation to the subjective symptoms met with in pericardial effusions, heart dilatation, etc.) He further showed that a heart which within the pericardial sac could be made to take 10.5 c.c. of fluid, when removed from the sac dilated to hold an additional 16 c.c., showing the support of the pericardium to the heart in dilatation. In pericarditis with effusion, the alterations of the pericardial tissue by the fluid and by inflammatory changes permit it to yield under the pressure of the accumulating exudate. If no adhesions form without, it returns to its normal size as the fluid is absorbed; but when it is fixed to the chest wall, the diaphragm, and other structures, as the fluid subsides it cannot return to its original dimensions, at least not entirely, and so the heart, whether inflamed or not, lacking any support from the outside, dilates as the fluid disappears until the two pericardial layers are again in contact. Furthermore, in a pericarditis with effusion, the heart and pericardium are separated by a considerable space over most of their surfaces. The heart cannot well become adherent until the fluid is absorbed, and if the pericardium is adherent externally adhesion between its two surfaces implies a dilatation of the heart to render this interpericardial adhesion possible. Now, rheumatic pericarditis is often a pericarditis with effusion, while the form of tuberculous pericarditis under consideration in this paper is presumably a simple plastic process, occurring as it does slowly and without symptoms. Hence the occurrence of cardiac incompetence often in one and seldom in the other. Myocardial degeneration and valvular lesions such as occur in rheumatism would, of course, favor the dilatation of the heart to a dangerous degree, from which it is ordinarily prevented by its own musculature. The chief difference between this theory and that of Dickenson is that *in the latter the dilatation of the heart is considered primary; in the former it is considered as secondary to the distention of the pericardium.*

Adhesions on the outer surface of the pericardium are most commonly thin filaments attaching it to the lung on either side, especially the left; such adhesions are of little importance, because of the elasticity of the lung. It is when the bands are firm and numerous, or the adhesion is of complete surfaces, and the pericardium is united to the chest wall, or to the lung, when it is in turn firmly united to the chest wall, or, perhaps most important of all, when the pericardium has spread over the diaphragm for a considerable distance, that the heart is affected. It is by no means unreasonable to consider that by the traction of these different points of attachment during inspiration, the diaphragm and the chest wall pulling in different directions, that a dilatation begun by the contraction of new-formed connective tissue would be increased. This traction during inspiration is accepted by many as the correct explanation of the pulsus paradoxus that is sometimes seen in the condition of pericarditis accompanied by mediastinitis. Of causes of simple plastic pericarditis with mediastinitis, other than rheumatism, pneumonia is the most prominent, for the inflammation, beginning outside of the pericardium, naturally leaves external adhesions. The simple plastic form of tuberculosis is probably seldom accompanied by much adhesion to the chest wall or diaphragm, unless occurring with a mediastinitis. J. H. Sequeira[70] has reported an interesting example of this condition occurring in an infant fifteen months old.

That adhesions limited to the pericardial cavity itself can so interfere with the action of the heart as to lead to asystole has been a muchdebated question. Many of the English writers are convinced that internal pericarditis alone, especially when of rheumatic origin, may do so. For example, A. G. Barrs[11] has reported four cases, all in young people, in which there were no adhesions to the anterior chest wall, and in one only a few adhesions to the lungs. Theo. Fischer,[12] writing on the importance of pericarditis in children, states that of a total of 196 cases of adherent pericarditis occurring in Guy's Hospital only eleven had adhesions to the anterior chest wall. As he found that in seven years' time, while there had been but four deaths due to valvular disease in children under fifteen years of age, there had been thirteen from adherent pericardium, it is evident that most of these fatal cases of adherent pericardium must have been without external pericarditis. Carl Hirsch[13] has made a valuable study of cardiac hypertrophy by ascertaining the ratio between the weight of the body and the weight of the heart and of its individual parts. He arrives at the conclusion that simple obliteration of the pericardium cannot bring about hypertrophy. This conclusion is, however, based on the findings in but six cases, without reference to the cause of the adhesions, nor to the existence of external adhesions. Dickenson[14] takes the same view, and states that in nearly all cases of rheumatic pericarditis there are adhesions externally, although in his own series of cases this condition was not marked in about half. However, it is to be remembered that in removing the sternum as ordinarily done at an autopsy, any abnormal adhesion of the pericardium anteriorly would be overlooked, as a rule, unless quite extensive.

An illustration of asystole, without external adhesions of consequence, is afforded by Case X. During life this was considered a case of broken compensation from valvular lesions. At autopsy the valves were found quite normal, the cavities dilated, walls hypertrophied, and the pericardial cavity was obliterated by strong adhesions without much thickening of the pericardial walls. There were a few adhesions between the left lung and the pericardium, but, although searched for, no other adhesions were found. Here, apparently, a fatal incompetence had resulted from simple internal adhesions. Macroscopically, the myocardium was unchanged.

How an internal obliterative pericarditis can interfere with the heart to any extent is difficult to see. Romberg[14] has suggested that the fibrous tissue, contracting, interferes with diastole, which supposition hardly agrees with the prominent dilatation in all these cases. Myocardial change in some instances is, perhaps, responsible, and this is sometimes to be distinguished only microscopically, as in a case reported by Jaccoud,[15] who considers that secondary changes are responsible for the incompetence of synechia more often than mere mechanical traction. In a number of cases that I have examined the myocardial lesions have been slight and limited to the tissues immediately beneath the pericardium. This applies also to the fatty infiltration which is often seen to cut out the superficial part of the musculature. The mere inelasticity of the adherent pericardium is not sufficient to cause any significant interference with cardiac action, and "there is abundant evidence that the mere fact of obliteration of the pericardial cavity around a normal heart is a trivial impediment to its action, and excites neither hypertrophy nor dilatation." (Dickenson.[18]) The fatal termination of the process produced by the pericarditis is not generally long delayed; often it is within a year of the beginning of the acute inflammation. In the case of Jaccoud,[23] previously referred to, death occurred about three and a half months after the onset.

An interesting and important condition which results from pericardial synechia is that of "pericarditic pseudocirrhosis" of the liver. This was first described by F. Pick[26] in 1896, and has attracted much attention since that time. He stated that the cases resembled much the mixed form of cirrhosis of the liver, with enlargement of the liver, and ascites, but without jaundice, occurring especially early in life. In all these cases the pericarditis is latent. The cause of the symptoms he considered as eminently a cirrhosis due to connective tissue proliferation resulting from obstruction in the circulation of the liver, and to intense ascites through portal congestion. R. C. Cabot,[27] in 1898, reporting a case, collected seventeen others from the literature, and the reports have increased at a corresponding rate since that time. V. Eisenmenger[28] most correctly objects to the relation between passive congestion and cirrhosis assumed by Pick, for little hyperplasia of connective tissue results from passive congestion in the liver, and that which does form is in the centre of the lobule. The ascites, without œdema of the legs, he attributes to conditions outside the liver, chiefly torsion, compression, or angulation of the inferior vena cava by the pericardio-mediastinal adhesions; or else through a localized peritonitis, with some possible etiological relation to the pericarditis, at the transverse fissure of the liver. This last is, perhaps, most marked in the perihepatitis or "zuckergussleber" of Curschmann. One interesting observation is the following: In seven of Diemer's[29] ten cases of calcification of the pericardium, in which the condition of the liver was described, atrophic cirrhosis was present; in an eighth there was extensive perihepatitis and syphilitic cirrhosis; in the ninth there was perihepatitis alone; in one only of the ten there was no cirrhosis, but simple passive congestion. Only one gave a history of alcoholism, and that but moderate. In two of the three cases of pericarditis in Pick's first report the exudate was calcareous, and in both of these there was a slight but demonstrable cirrhosis of the liver; the third, in which the adhesions were not calcified, showed no cirrhosis. C. Bacaloglu[30] reports a case of calcareous pericardium, not included among those of Diemer, showing a slight atrophic cirrhosis; no history given. In all four cases of calcification of the exudate in the writer's series a marked atrophic cirrhosis was present, although without ascites. Of these four, one gave a history of alcoholism (Case XVI.), but the other three were not alcoholics. From a study of the autopsy reports and history-sheets of these cases I am unable to ascribe any relationship between the pericarditis and the cirrhosis; but the coincidence is striking. However, it may be said that *while in fibrous pericarditis we do get a symptom-complex entitled to the name of pericarditic pseudocirrhosis, in calcareous pericarditis we frequently, in the great majority of reported cases, in fact, get a real atrophic cirrhosis.* This association of cirrhosis is not seen in cases of simple fibrous pericarditis. A case reported by Ewart,[11] of calcareous pericarditis, presented marked œdema and conditions leading to the diagnosis of atrophic cirrhosis, which did not exist. In this case the operation for establishment of collateral circulation by producing plastic peritonitis was performed.

An individual with both cardiac and tuberculous lesions may present them combined in an interesting form in the liver, as the so-called *cardiotuberculous liver.* This is simply a marked chronic passive congestion of the liver, plus tuberculous changes, which may be, according to Cousin,[30] either specific tuberculous lesions, or fibrous or fatty changes with or without tubercles. Tuberculous pericarditis offers the proper conditions for the development of this form of hepatitis, and it has been seen in many cases. Hutinel[11] has reported several cases in children, and considers the subject extensively.

W. H. Welch[31] has written of *thrombosis of the systemic veins* occurring in cardiac disease. In twenty-seven such cases collected by him the pericardium was found adherent three times, all being rheumatic. This has been observed in two of my series (I. and XI.); in both of these and in four others (V., VI., VIII., and XIII.) there were thrombi in the auricles. The snaring off or immobilization of parts of the auricles, particularly the appendix, furnishes most favorable conditions for thrombus formation, and embolism from this source is frequent. This is, therefore, a dangerous complication. Welch ascribes the thrombosis of the systemic veins in part to the eddying or whirling movement of the blood that may be set up in heart disease in the lower end of the jugulars and the adjacent parts of the innominate and subclavian veins, in part to the anatomical disposition and structure of the veins, but is inclined to think that infection is the important factor. In his first case, which resembled Case XI. of my series, in almost every detail, streptococci were found in the thrombus. In Case XI. the thrombus in the left axillary vein contained staphylococci, both aureus and albus, also the bacillus coli communis, and a small, strictly anaërobic organism, the exact nature of which was indeterminate; it stained by Gram's method. From the heart's blood only S. pyogenes aureus and B. coli communis were obtained. The other case of thrombosis (Case I.) yielded S. pyogenes albus in cultures from the thrombus, which was not obtained in cultures from the heart's blood.

TERMINATIONS. It is quite probable that fibrous adhesions when once formed in the pericardial sac are not necessarily permanent. It is known that they are often removed from the peritoneal cavity in course of time, and this is probably true of the pericardial cavity. As the new-formed connective tissue becomes older and assumes its fibrillar structure the inevitable contraction that follows obliterates most of the bloodvessels, as in all scars. To this is added the incessant tugging of the heart, drawing them out and causing them to finally separate and form simple tags on the surface of the pericardium. This conception is formed from the frequent finding of the various stages of this process. As a rule, the parts of the adhesions that remain longest are those over the surface of the right auricle and also between the parietal layer and the apex and ventricular septum. Sibson[23] states that the reason for this is that these are the portions of the front of the heart which move least. As the "tugging" effect, as well as the interference with circulation by contraction, would be manifestly less in a completely obliterated sac than in isolated adhesions, the chances of complete absorption of adhesions will vary inversely with their extent, other conditions being equal. A healthy and strong heart would, of course, most readily free itself from the bands. Calcification is another possible termination.

The fibrous adhesions may also act as a "locus minores resistientiæ." Rubino[24] has shown the effect of injury to the pericardium on localization by first injuring the pericardium by heat, mechanically, and by severe cooling, and then injecting pus microbes into the general circulation. In this way he produced experimentally a pericarditis, which is the result that would be expected from what we know of the general laws of bacterial localization. That fibrous adhesions may produce in a slighter degree the same effect is not improbable, and it is not uncommon to see an acute pericardial inflammation engrafted upon a chronic adherent process in which the pericardium is but partly obliterated. On the other hand, total obliteration of the cavity would render secondary infection very unlikely to occur. McPhedran and Caven[25] in reporting a case of diffuse tuberculous hepatitis state their opinion that a coexisting pericarditis was implanted in this way on an older fibrous pericarditis.

REFERENCES.

1. Loudet. Quoted by Whittaker, Twentieth Century Practice, vol. vi. p. 80.
2. Mracek and Genersich. See correspondence from Budapest in Lancet, January 23, 1897, p. 241.
3. F. v. Recklinghausen. Virchow's Archiv, 1885, vol. c. p. 503.

4. W. C. F. Witte. Philadelphia Medical Journal, May 7, 1898.
5. Roberts. Allbutt's System, vol. vi. p. 729.
6. M. Labbé. Bull. d. l. Soc. Anat., 1897, p. 246.
7. Kast. Virchow's Archiv, 1884, xcvi., p. 489.
8. Mickle. Quoted by Hayem and Tissier, Rev. d. Med., 1889, vol. ix. p. 41.
9. A. N. Perón. La Presse Méd., February 19, 1898, p. 93.
10. J. Auclair. Arch. Méd. Exper., 1900, vol. xii. p. 189.
11. J. B. Herrick. Chicago Med. Recorder, 1898, vol. xiv. p. 192.
12. F. J. Bose. La Presse Méd., September 28, 1898, p. 185.
13. W. Ewart. Lancet, November 21, 1896, p. 1446.
14. Fritz Diemer. Zeitschr. f. Heilk., 1899, vol. xx. p. 257.
15. Drummond. AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1890, vol. xcix. p. 153.
16. Püschmann. Inaugural Diss., Leipzig, 1896. Abstract in Centr. f allg. Pathol., 1897, vol. viii. p. 818.
17. Wm. Ewart. Med. Press and Circular, April 19, 1899, p. 400.
18. W. L. Dickenson. AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1896, vol. cxii. p. 691.
19. H. L. Barnard. Journ. of Physiology: Proceedings of the Physiol. Society, 1898, vol. xxii. p. xiii.
20. J. H. Sequeira. Lancet, 1898, vol. ii. p. 1765.
21. A. G. Barrs. Lancet, September 17 and 24, 1881, pp. 500 and 544.
22. Theo. Fischer. Bristol Med.-Chir. Journ., June, 1894.
23. Carl Hirsch. Deut. Arch. f. klin. Med., 1900, vol. lxviii. p. 321.
24. Romberg. Quoted by Hirsch, loc. cit.
25. Jaccoud. Sem. Méd., 1893, vol. xiii. p. 21.
26. F. Pick. Zeits. f. klin. Med., 1896, vol. xxix. p. 385.
27. R. C. Cabot. Boston Med. and Surg. Journ., 1898, vol. cxxxviii. p. 463.
28. V. Eisenmenger. Wien. klin. Woch., 1900, vol. xiii. p. 249.
29. C. Bacaloglu. Bull. Soc. Anat. de Paris, 1899, p. 68.
30. Cousin. Gaz. heb. d. Méd. et. d. Chir., January 14, 1900.
31. Hutinel. Rev. des Mal. d. l. Enf., 1893, vol. xi. p. 529, and 1894, vol. xii. p. 15.
32. W. H. Welch. Festschrift in honor of A. Jacobi, 1900, p. 463.
33. Silson. Quoted by Roberts, loc. cit.
34. Alfredo Rubino. Arch. Ital. de Biol., 1892, vol. xviii. p. 298. See Schmidt's Jahrb., vol. ccxlvii. p. 191.
35. A. McPhedran and John Caven. AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1893, vol. cv. p. 511.

A CASE OF FIBROMA MOLLUSCUM.

By M. B. Hartzell, M.D.,

INSTRUCTOR IN DERMATOLOGY, UNIVERSITY OF PENNSYLVANIA; DERMATOLOGIST TO THE METHODIST HOSPITAL OF PHILADELPHIA AND TO THE PHILADELPHIA HOSPITAL.

In July of this year, G. J., a young, unmarried woman, aged twenty-six years, came to the Skin Dispensary of the University Hospital for advice concerning a disease of the skin characterized by the presence of a large number of variously sized tumors distributed over the entire cutaneous surface. These tumors were covered by normal skin, and varied in size from a hempseed to a pigeon's egg; they were soft and inelastic to the touch, the largest feeling like empty sacs, and, according as they were small or large, they were hemispherical and sessile, or pear-shaped and pedunculated. While all parts of the skin were affected, the tumors were most abundant and largest upon the arms. The history of the affection, very briefly, was that the tumors began to appear in infancy—it was asserted most positively by other members of the family that none were present at birth—and rapidly increased in number, new lesions continuing to appear up to the present time. Her general